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EDITORIAL.

THE enigma of "ether convulsions" remains unsolved. In spite of the well argued explanation, an alkalosis due to acapnia, which Dr. W. W. Kemp advances in the article which we publish later, it is plain, even on his own showing there, that the causation which he demonstrates is not true of all cases. There are certainly facts in the history of this phenomenon which appear to lend support to Dr. Kemp's theory. Ether convulsions were unknown, so far as we can discover, during those years when ether was given solely by closed or semi-closed methods. Yet during that period, the reign of the Clover and the Ormsby, many thousands of patients were put deeply under the influence of ether. None of them were, of course, subjected to the least CO_2 deprivation. Very much the opposite. Many were rendered cyanotic both by excess of CO_2 and by lack of oxygen. Yet ether convulsions were notably absent. So far the history certainly favours Dr. Kemp. Open ether, however, first described about 1895, was widely used at least from 1907 to 1922. A vast number of individuals must have been subjected to this process during those years. Nevertheless, no ether convulsions are recorded till about 1913. It is hard to believe that in all that time not one subject was exposed to the influence which Dr. Kemp claims as causative, yet we hear of no ether convulsion. What is the explanation? We

confess ourselves at a loss to supply one. It is a curious fact that since instances of ether convulsions have been recorded the phenomenon has appeared during both forms of ether anæsthesia, the closed and the open. They have certainly been much less frequent with the former method, but that method is now by far the more rarely employed. At first it looked as if they were especially apt to occur when oxygen was driven through ether, but that contention has not been upheld by subsequent events. Our understanding of this strange and objectionable phenomenon has not sensibly advanced since Dr. Charles Hadfield's analytical paper, in which the only factor he could pin down as apparently common to all cases was that of warmth!

UNUSUAL COMPLICATIONS OF SURGICAL ANÆSTHESIA UNDER ETHER

By W. N. KEMP, B.A., M.D.
Anæsthetist, Vancouver General Hospital, Vancouver, B.C.

IT is my purpose to discuss six case histories which illustrate two types of unusual complications of surgical anæsthesia under ether. In the first series of three cases, the so-called deep ether convulsions were the complicating phenomena; in the second series of three cases, unexpected cardiac arrest with temporary resuscitation are the interesting features.

SERIES I.—CONVULSIONS DURING SURGICAL ANÆSTHESIA UNDER ETHER.

Case I., Mr. A. V. This man, a Russian Jew of 49 years, was admitted to the Vancouver General Hospital on February 14, 1932, as an abdominal emergency case. A laparotomy under semi-closed ether vapour-with-oxygen anæsthesia was immediately performed and a perforated gastric ulcer was sutured. Thirty-five minutes after the induction of anæsthesia while the surgeon was closing the peritoneal cavity, muscular twitchings started in the facial muscles and soon extended to the arms and abdominal muscles. On the predetermined theoretical assumption that these muscular contractions were due to carbon dioxide loss and the consequent alkalosis thus established in the blood, the patient was forced to rebreathe into the bag of the gas machine into which plenty of oxygen was passed. Within a few minutes the convulsions ceased. Ten minutes later at the close of the operation a catheter specimen of urine was taken and examined for reaction. The alkalinity of this specimen of urine confirmed the alkalosis of the blood during the convulsions. Subsequent specimens of urine were acid in reaction.

Having made an uneventful recovery from the laparotomy, this patient was again anæsthetized seven weeks later so that a partial gastrectomy might be performed. Intratracheal oxygen-ether anæsthesia was used. Within 20 minutes of

the introduction of the tracheal catheter, twitchings of the facial muscles were noticed. By immediately converting the " open " intratracheal anæsthesia into a " closed " intratracheal anæsthesia (by covering the patient's face with a mask and damp cloths), his carbon dioxide was conserved and rebreathed and the twitchings ceased. They could be re-established at will by removing the face mask and allowing the carbon dioxide to escape. The patient made a good recovery, and was discharged from hospital one month later.

Case 2, Mr. A. H. This patient, an Englishman of 22 years, was anæsthetized on July 28, 1932, for appendectomy. The usual ethyl chloride-ether sequence was used to induce anæsthesia, the latter being maintained with oxygen-ether vapour under a face mask. Twenty minutes after the induction of anæsthesia and at the completion of the actual appendectomy, convulsive movements of the face, arms, and abdominal muscles occurred. These were immediately controlled by forcing the patient to rebreathe into the bag of the gas machine with extra oxygen added. He made an uneventful recovery and was discharged from hospital 12 days later.

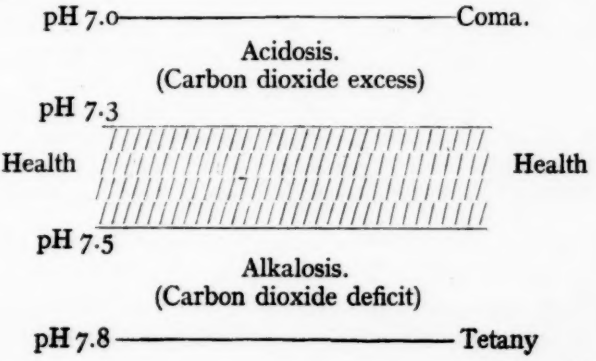
Case 3, Child I. I. This Japanese boy of nine years was admitted to hospital on November 15, 1932, with a diagnosis of acute appendicitis. The appendix was removed under semi-open ether-oxygen anæsthesia. About 25 minutes after induction of anæsthesia, as the surgeon was suturing the peritoneum, marked clonic movements of the muscles of the face, the arms and the abdomen occurred. Unlike the two preceding cases, in this patient the convulsive attack was not preceded by pulmonary over-ventilation, nor was it controlled by the rebreathing technique. Chloroform by the drop method with oxygen insufflation of the pharynx stopped the convulsions, and the child returned to the ward in good condition. He made an uneventful recovery.

Discussion.

Convulsions during ether anæsthesia have been reported with increasing frequency during the past decade, especially in the British journals. Many and varied theories have been

suggested to explain this serious complication of ether anæsthesia. Time does not permit a consideration of these theories here. In 1932 I advanced the "Acapnia Theory" (I, 2) to explain the majority of deep ether convulsions. Since then further successful experience in the carbon dioxide or rebreathing treatment in a total of five cases has established the soundness of the theory and treatment in cases of convulsions preceded by pulmonary over-ventilation. One meets with an occasional case (as Case 3) where there is no preceding over-ventilation and where chloroform is our sheet anchor.

A brief résumé of the "Acapnia Theory" will be attempted here. The most fundamental constant of human life is the hydrogen ion concentration (cH) of the blood. The exponent that expresses the H-ion concentration is abbreviated to pH. In health the human H-ion concentration may vary from pH 7.3 to pH 7.5. As Rowntree[3] has aptly phrased it, this means a variation from the "acidity" of distilled water (pH 7.3) to the "alkalinity" of tap water (pH 7.5). Still compatible with life, but incompatible with health, we have an upper (alkaline) limit of cH as it is expressed by the exponent pH 7.8. This condition of cH is called *alkalosis* and is manifested clinically by *tetany*. The lowest limit of cH compatible with life is expressed as pH 7.0. This condition of cH is called *acidosis* and its clinical manifestation is *coma*. Graphically this range of cH may be represented thus:—



For many years Prof. Yandell Henderson has taught that the only *immediate* regulation of blood cH is accomplished by respiration. There exists a constant relation between the free carbon dioxide of the blood plasma and the blood alkali which is expressed by the following equation:

$$\frac{CO_2 + H_2O}{\text{Blood Alkali}} = \frac{H_2CO_3}{NaHCO_3} = \frac{3}{60} = \frac{1}{20}$$

The numerator in the ratio $\frac{1}{20}$, representing, as it does, carbon dioxide, in simple solution in the blood plasma, can be quickly increased or decreased by slowing or quickening pulmonary respiration in order to prevent any upset in the normal 1:20 ratio from a quantitive change in the denominator (representing the blood alkali). This respiratory balancing of the CO_2 is an emergency measure to maintain the normal 1:20 acid-base balance or the normal H-ion concentration. Another important but slower physiological step in maintaining this normal 1:20 acid-base ratio, is the elimination or retention of alkalies and ammonia by the kidneys. In this way compensatory quantitative adjustments have been made.

It is obvious that the rapid elimination of CO_2 by some artificial stimulation of the respiratory centre such as is produced by etherization and operation will upset the normal 1:20 acid-base ratio and tend to produce a blood alkalosis. Before this ratio can be restored to normal by elimination of alkali via the kidneys, an alkalosis of sufficient degree (pH 7.8) to produce tetany may occur. Patients suffering from peptic ulcer who have been on an alkaline diet may, by over-ventilation, be easily thrown from a state of compensated alkalosis into a state of uncompensated alkalosis and tetany. Theoretical considerations and practical treatment of ether convulsions indicate that acapnia or carbon dioxide deficit is the causative factor in many instances.

Series 2.—Unexpected Cardiac Arrest During Ether Anæsthesia With Temporary Resuscitation.

In the past four years at the Vancouver General Hospital, there have occurred on the operating table three cases of

definite cardiac arrest and apparent death, in each of which life was temporarily restored by methods to be described later.

Case 1. Child R.P. In 1929, this patient, a boy of six years, was easily induced with ethyl chloride-ether anæsthesia in preparation for tonsillectomy. Anæthesia was maintained during operation by intrapharyngeal oxygen-ether vapour. Forty minutes after the induction of anæsthesia while the operator endeavoured to secure hæmostasis in the second tonsillar fossa, the child collapsed suddenly. Various resuscitative measures were adopted, but were barren of result until 15 minutes later, when all hope had been given up, the heart beat and respirations were restored while the Sylvester method of artificial respiration was being employed. So satisfactory did his general condition become, that, the bleeding point in the left tonsillar fossa was ligated and the adenoids were removed. He was returned to the ward with a pulse of 90 and apparently in excellent physical condition. He became cyanosed shortly after returning to the ward and developed a series of convulsions in one of which he died 14 hours later. A clinical diagnosis of œdema of the brain was made and unfortunately no autopsy was held.

Case 2. Miss A. C. This young white woman of 22 years in apparent excellent physical condition was anæsthetized by the usual ethyl chloride-ether sequence in preparation for tonsillectomy. This operation was successfully performed under intratracheal oxygen-ether vapour when the patient suddenly collapsed and became pulseless. Artificial respiration and attempted intra-cardiac injections of 1 c.c. of 1/1000 solution of adrenalin chloride appeared to be without result when, 20 minutes later, her pulse, normal colour and respirations gradually returned. After an hour's stay in the operating room, during which time she received 1000 c.c. of normal saline, she was sent to the ward apparently in good general condition. After her return to bed she was in collapse for a short time. When she rallied from the collapse, she had a convulsion. On the assumption that edema of the brain was the etiological factor in her clinical condition, dehydration therapy in the form of 30 per cent NaCl solution was used intravenously. This and other forms of

supportive treatment were without lasting effect and the patient died ten hours after returning to bed.

Post-mortem revealed nothing of note except a collapsed left lung and a thymus gland that weighed 23 grams.

Case 3. Miss K. O'G. This patient, a girl of 16 years, apparently in good general condition except for an attack of acute appendicitis, was anæsthetized in January, 1933, with ethyl chloride and ether for appendectomy. Under intratracheal oxygen-ether vapour, an acutely inflamed retrocæcal appendix was removed with difficulty. With the exception of a brief period of so-called deep ether convulsions, which were readily stopped by the rebreathing technique discussed earlier in this paper, her anæsthetic course was normal until about one hour after the induction when a degree of cyanosis was noted. This cyanosis got progressively worse in spite of free natural pulmonary ventilation with straight oxygen intratracheal insufflation. Within ten minutes of the onset of cyanosis the heart stopped beating (confirmed by intra-abdominal palpation). Immediate resuscitative measures were promptly instituted, including artificial respiration with forced carbon dioxide (5 per cent) and oxygen inhalation and intracardiac injection of 1 c.c. of adrenalin chloride 1/1000 solution. It was not until intravenous saline was successfully introduced and 6 minims of adrenalin chloride 1/1000 was injected into the rubber tubing of the intravenous set that any cardiac response was elicited. Then, 15 minutes after cardiac arrest, the heart commenced to beat forcibly and regularly and with artificial respiration and carbon dioxide-oxygen forced inhalation, her skin colour became pink. Strange to say, normal respiratory function did not occur until 45 minutes later and appeared to have been uninfluenced by intravenous strychnine (1/100 gr.) or alpha-lobulin (1 ampoule of 1 c.c.). When this patient left the operating room for the ward her general condition appeared to be that of a normal post-operative case. After returning to bed her general condition changed markedly for the worse. Convulsions and depressed circulatory function featured her post-operative condition. She died ten hours later in spite of supportive treatment. The pathologist's report was "no

anatomical cause of death found." The only unusual finding was a thymus gland of 25 grams.

Discussion.

The easiest and perhaps the most natural explanation that can be offered to explain these unusual cases of cardiac arrest and resuscitation on the operating table is to the effect that these patients suffered from a simple overdose of ether, were subsequently revived with difficulty, and died a few hours later as a direct result of the prolonged period of cerebral anoxemia from which they suffered before the heart beat was restored. The fundamental fallacy of this explanation is that, as all experienced anæsthetists know, *the first reaction of an animal or human to an overdose of ether is respiratory cessation*. In the cases under discussion, cyanosis in the presence of a free airway, adequate oxygen supply and spontaneous respiratory function was the first untoward sign. In experimental animals the heart continues to beat for several minutes after ether overdosage. In humans also there is a wide margin between overdosage with respiratory cessation and cardiac arrest. If it were not so, fatalities on the operating table would be a very common occurrence indeed.

In explanation of these tragic events one must fall back to hypothetical grounds. I would suggest that, although these unfortunate individuals did not die from ether overdosage in the usually accepted sense, yet they did die from ether overdosage in as much as time is a factor in overdosage. To them a surgical anæsthesia lasting 40, 30, or 60 minutes respectively, is a "time-ether" overdose. A perfectly normal person might withstand anæsthesia for several hours; the time that these unusually constituted individuals can withstand anæsthesia is to be measured in minutes rather than hours.

This varying degree of ability to withstand anæsthetic and operative shock would seem to be analogous to the variation in individual resistance to infection often noticed by physicians. In at least three well known endocrine dysfunctional conditions, *hypothyroidism, diabetes mellitus,* and *Addison's disease* there is a marked lowering in the individual's infection resistance. Indeed, one might reasonably suppose that lack of resistance to both infection and anæs-

thesia in certain apparently normal persons may be due to an unsuspected hypofunction of important endocrine glands such as the adrenal cortex and the thyroid gland.

That there is an endocrine basis to one's ability to withstand anæsthetic and operative shock is strongly suggested by the recorded observations of many workers in the realm of animal experimentation. It has been reported by Marine[4] that the majority of dogs in Cleveland, Ohio, have goitre and are poor anæsthetic risks. F. Hartman[5] has reported that a cat, after total adrenalectomy, but while kept in apparent normal condition by daily injections of *cortin* (a potent extract of the adrenal cortex first isolated by Dr. F. Hartman), proved to be a poor operative risk, dying under skilled ether anæsthesia of only a few minutes' duration. It has been frequently noted[6, 7] that animals with a complete or partial cortico-adrenal insufficiency are prone to develop respiratory and cutaneous infections. These observations suggest that, in animals, *lack of resistance to infection and to anæsthesia may be due to similar endocrine hypofunction.*

Recent research work in cortico-adrenal insufficiency by Firor and Gollman[8] shows that rats, like all other animals, cannot survive a *complete* bilateral adrenalectomy. (It has been repeatedly shown that the death of adrenalectomiged animals is due to loss of the vital cortex of the adrenal gland and is not at all related to the loss of the adrenine secreting medulla.) Firor and Gollman showed that the survival time of adrenalectomized rats is directly controlled by (*a*) preoperative care, (*b*) the skill of the operator, (*c*) the duration of ether anæsthesia, and (*d*) the post-operative care.

A careful review of recent endocrine research suggests that there is a very important direct relationship between *the adrenal cortex, the thyroid and thymus glands, and general lymphoid hyperplasia.* The clinical evidence to support this postulated relationship is as follows:—

(*a*) Young adults suffering from Addison's disease (now definitely known to be due to aplasia or disease of the adrenal cortex) often show, on autopsy, enlargement of the mesenteric lymphatic glands and the thymus gland.[9, 10]

(*b*) Young adults suffering from Graves' disease commonly show, on autopsy, hyperplasia of the lymphoid tissues including the thymus gland.[11, 13]

(*c*) Young adults suffering from either Graves' or Addison's disease usually show a blood lmyphocytosis as high as 60 per cent.

(*d*) Recent clinical investigations by Crile and associates indicate that there exists a functional relationship between the thyroid gland and the adrenal cortex.

The experimental evidence in favour of this postulated cortico-adrenal-thyroid-lymphoblastic tissue relationship is very strong:—

(*a*) Bilateral two-stage adrenalectomy in dogs results in the death of the animals with typical signs of cortico-adrenal insufficiency; in every case post-mortem examination reveals marked enlargement of the mesenteric lymphatic glands and of the thymus gland.[13]

(*b*) It has been shown by Jaffe[14] that the thymus of rats regenerates after adrenalectomy. Marine[15] and associates have shown that, in the rat (1) thyroidectomy hastens the involution of the thymus gland, and (2) adrenalectomy causes regeneration of the thymus gland and the lymphoid tissues.

(*c*) Marine[16] has shown that, in the rabbit and cat with intact thyroid gland, a transient symptom-complex which closely resembles Graves' disease can be produced by sufficient but sub-lethal injury to the adrenal glands. The outstanding symptoms are increased metabolism, myasthenia, increased appetite, increased irritability and *increased susceptibility to drugs*. These symptoms are accompaniel by *regeneration of the thymus gland* and *hyperplasia of the lymph glands*.

(*d*) Partial cortico-adrenal insufficiency operatively established in dogs is compatible with apparent health (with lowered resistance to infection); the post-mortem examination of such experimental dogs shows pathological findings

strikingly similar to those found in cases of status lymphaticus, Graves' disease or Addison's disease, viz., ***enlargement of the thymus gland and of the mesenteric lymphatic glands.***[7]

The foregoing clinical and experimental evidence indicates that there is a definite relationship between the adrenal cortex on the one hand, and thymus and lymphoid hyperplasia on the other. The thyroid gland also is related to cortico-adrenal dysfunction and also to lymphoblastic hyperplasia. It appears from the clinical evidence of Graves' and Addison's disease that dysfunction of either (or both ?) the adrenal cortex or the thyroid gland in young alults most *commonly results in enlargement of the thymus gland and hyperplasia of the mesenteric lymphatic glands.* Conversely, it may reasonably be assumed that the post-mortem finding of enlargement of the thymus gland and the ideopathic hyperplasia of the mesenteric lymphatic glands indicates, *ipso facto*, the pre-existence of dysfunction of either or both the adrenal cortex and the thyroid gland.

It is common knowledge that, without special preoperative preparation, patients with that form of thyroid dysfunction commonly known as hyperthyroidism are decidedly poor operative risks even with local or gas-oxygen anæsthesia. Likewise, the victims of Addison's disease are poor risks with any form of anæsthesia. *It follows, therefore, that the post-mortem finding of enlargement of the thymus gland or ideopathic hyperplasia of the mesenteric lymphatic glands indicate that the individual in question was handicapped by a partial dysfunction of the adrenal cortex or the thyroid gland or both and with such a handicap was not able to withstand the " time-shock " factors of anæsthesia and operation.*

Cardiac Resuscitation.

The consideration of the foregoing cases of cardiac arrest on the operating table, naturally raises the question of the methods and value of cardiac resuscitation. This question will be discussed under the following heads:—(*a*) Intra-cardiac injection, (*b*) cardiac massage, (*c*) intravenous therapy, (*d*) intra-arterial perfusion with calcium chloride solution.

(*a*) *Intracardiac Injection.* The direct intracardiac injection of stimulating drugs was first suggested by Winter[17] in 1905 on the strength of his animal experimentation. In 1923 Bodon[18] reported a case and reviewed the literature. Up to that date 90 cases of attempted intracardiac injection had been reported, in only 24 of which was the heart-beat restored. Hyman[19] in 1930 published some important conclusions regarding this method of resuscitation which were based on clinical research. They are: (1) Intracardiac injection should be reserved for the normal heart which has stopped. (2) No medication is necessary in the so-called "injection," the stimulation of the needle alone being efficacious in favourable cases. (3) The needle should be inserted into the auricle to get the best results; here it acts as a focus of extra systoles which serves as a temporary pacemaker in the auricular musculature. (4) Positive results are to be expected in only 25 per cent of cases. (5) The auricle is reached without endangering the great vessels by inserting the needle in the third interspace to the right and close to the sternum ($3\frac{1}{2}$ to $4\frac{1}{2}$ inches depth in adults, 2 inches in children).

(*b*) *Cardiac Massage.* Bodin[18] (1923) quotes Pieri's statistics concerning cardiac massage in resuscitation. In the latter's series of 76 cases from the literature, only 20 were successfully revived by cardiac massage. In 23 cases a thoracic exposure was made; in 12 a transdiaphragmatic exposure was used; in 41 cases sub-diaphragmatic massage was used. Bodon does not state in which type of massage the best results were obtained. Bailey[20] in 1927 compared his experience with intracardiac injection and cardiac massage to the advantage of the latter.

(*c*) *Intravenous Medication.* It is obvious that intravenous medication can be of no value in cases of cardiac arrest unless it is carried to the heart in an infusion of normal saline or some other isotonic solution. There is no doubt that an intravenous infusion of normal saline carrying 6 minims of adrenalin chloride 1/1000 solution started the heart (in the third case in our second series above mentioned) after an accurate (as shown by post-mortem) intracardiac injection of adrenalin chloride (into the left ventricle) and after sub-diaphragmatic cardiac massage had failed.

(*d*) ***Intra-arterial Perfusion with Calcium Solution.*** In a previous paper[21] the opinion was expressed that in cases of sudden cardiac arrest the intra-arterial perfusion of the heart with normal saline containing adrenalin chloride should be attempted because such therapy was based on sound principles of cardiac physiology. Since then this opinion has been strengthened and the suggested *modus operandi* slightly altered by subsequent experimental work and by a consideration of the pioneer work of Crile and Dolley[22] in 1906 and the more recent works of Hooker[23] in 1929. The former workers employed intra-carotid therapy in dogs whose hearts had ceased to beat (as judged by the absence of peripheral pulse and heart sounds) following a deliberate overdose of chloroform or ether. Having tried (*a*) cardiac massage (*b*) cardiac massage with artificial respiration and intravenous infusion of saline containing adrenalin chloride, with only limited success, they resorted to the perfusion of Ringer's solution into the central end of the carotid artery under pressure of 30 to 40 mm. Hg. (such pressure having been reported as necessary to normal cardiac activity). To the Ringer's solution was added adrenalin chloride and hirudin, the purpose of the latter being to prevent intravascular clotting. The perfusion was accompanied by artificial respiration and rhythmic thoracic compression. Crile and Dolley reported that, under the above conditions, dogs who showed no signs of cardiac activity for five minutes *were uniformly and readily resuscitated;* after ten minutes consciousness was rarely restored, while after 23 minutes in adult dogs and 35 minutes in puppies, complete return of circulation could not be accomplished by this method.

While working on the problem of ventricular fibrillation following electric shock, D. R. Hooker[23] showed that the fibrillating dog heart can be completely arrested by the carotid perfusion of 0.5 per cent solution of KCl and that *normal cardiac function can be subsequently restored by the carotid perfusion of* 0.023 *per cent solution of calcium chloride in normal saline,* with subsequent recovery of the dog. Hooker's procedure consists of the central carotid injection of the remedial solution, saturated with oxygen and warmed to body temperature *at a pressure of* 150 *mm. Hg.*

When the heart is stopped and the arterial pressure practically at zero, the calcium solution readily finds its way into the coronary arteries and the cardiac musculature. The resuscitating solution (using 25 cc. per kilo.) washes the excess KCl (used in the production of cardiac inhibition) out of the coronary bed and substitutes a relative excess of the contraction stimulating ion, calcium. In the experience of Hooker this resuscitative effect is enhanced by the coincident injection into the perfusion fluid of 1 cc. of 1 : 1000 adrenalin chloride solution.

Concerning "Resuscitation of the Heart *in situ*," Prof. J. J. R. Macleod[24] states in his most recent text-book: "A suitable intra-coronary pressure is a *sine qua non* for the maintenance of the heart beat, and this is a fact of great clinical significance, for it indicates that any attempts to resuscitate a dead animal are certain of failure unless the method is such as will bring a nutrient fluid under a certain pressure to bear on the coronary arteries. Injection of fluid, even of defibrinated blood, into a vein will obviously fail to fulfil this condition, for the perfusion must be made into an artery so that the fluid is carried down the aorta and thence to the coronary arteries."

Experimental perfusion of the mammalian heart outside the body shows that, in addition to an optimum temperature, pressure and oxygen supply, a definite specific concentration of the ions of sodium and calcium are absolutely essential to any cardiac function. In the cases of sudden cardiac arrest on the operating table, it is quite possible that some fundamental change in blood chemistry resulting from ether anæsthesia in a person of potential or actual endocrine imbalance, was the primary cause of the stoppage of the heart. It is known that a comparatively slight change in the H-ion concentration of the blood as caused by 10 or 15 minutes of pulmonary over-ventilation with oxygen will cause remarkable neuro-muscular phenomena in the form of tetany.[25] In view of the above mentioned clinical and experimental facts it is not unreasonable to postulate that *individuals of potential or actual endocrine imbalance (relating principally to the cortico-adrenal and thyroid functions) may die suddenly under ether or chloroform anæsthesia from some (as yet) un-*

proven, but possible, change in H-ion concentration or calcium-sodium concentration of the circulating blood.

Discussion of Resuscitative Measures.

The combined experiences of surgeons reporting the use of intra-arterial injection or cardiac massage show that the best odds in favour of cardiac resuscitation offered by either of these procedures are one chance in four. Experimental results in animals would indicate great possibilities for the intra-arterial perfusion of 0.023 per cent solution of calcium chloride in normal saline in individuals without cardiac pathology who die suddenly and unexpectedly on the operating table under general anæsthesia. In practical form this suggested resuscitative therapy consists of adding 1/4 c.c. of saturated CaCl solution to 1000 cc. of normal saline of body temperature and perfusing the coronary arteries by way of the brachial or carotid artery, using 25 c.c. per kilo. of body weight. To secure the necessary 150 mm. Hg. pressure the burette containing the solution must be elevated 6.5 feet above the heart level. The bubbling of oxygen through the perfusion solution in the burette is also indicated. The hypodermic injection of 1 cc. of 1 : 1000 adrenalin chloride solution into the tubing of the perfusion set is also called for. I would suggest the adequate testing of this suggested mode of cardiac resuscitation on persons dying of some hopeless chronic disease in the public wards or, as a last resort, in cases dying suddenly upon the operating table.

Summary.

In the first part of this paper convulsions under ether anæsthesia are discussed and practicable therapy is outlined, namely, carbon dioxide rebreathing. In the second part of the paper cases of unexpected cardiac arrest on the operating table are presented and discussed. The view is expressed that these persons die on the operating table under general anæsthesia by reason of some unsuspected endocrine dysfunction probably directly related to the adrenal cortex and the thyroid gland; the only *gross* anatomic manifestations of this dysfunction being hyperplasia of the thymus gland or the lymphoid glands, or both. In the third part of the paper

methods of cardiac resuscitation are discussed briefly with especial emphasis on a newly-suggested (clinical) method of so-called "intra-arterial perfusion."

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KIDNEY DISEASE AND ANÆSTHETICS

By R. J. Minnitt, M.D.

Hon. Anæsthetist The Royal Infirmary and The David Lewis Northern Hospital, Liverpool.

IN eclampsia, the pre-eclamptic state and in bladder diseases (especially of the prostate) kidney damage may be extreme, and therefore the subject of anæsthesia for such patients is of very great importance.

It is well known that the effect of an ether anæsthetic on renal function is considerable. As far back as 1899 and 1900 R. C. Kemp and W. H. Thompson, in America, and B. W. Buxton and A. G. Levy, in this country, were putting forward theories to account for the albuminuria after ether anæsthesia. In 1927 Haines and Milliken[1] examined between 60 and 70 cases, and showed that when ether was administered albumin was present in the post-operative urine. It appears, therefore, that ether produces transient renal damage, probably slight cloudy swelling. Florence Stephen[2] has similarly reported albuminuria in all of a series of cases examined after ether anæsthesia, and has suggested that the anæsthetic causes changes in the renal cells.

In the accompanying table, extracted from a number of cases as an illustration, there are given urea estimations before and after anæsthesia, and 24 and 48 hours later. It should be noted that no change takes place in the urea during the time of anæsthesia, but the table suggests two things: (1) The higher the urea content of the patient, the greater the rise in the day or two following the operation; (2) There was no abnormal change in the blood-urea on the days following administration of gas and oxygen.

As an illustration of what happens in prostate cases, mention is made of a patient who had a one-stage prostatectomy under gas and oxygen anæsthesia, with the addition of 3 oz. of ether, the time taken being three-quarters of an hour.

COMPARISON OF UREA ESTIMATIONS IN RELATION TO ANÆSTHESIA

Operation	Method	Amount of ether used	Time of anæsthesia	Before and after op. mg.%	24 hours after mg.%	48 hours after mg.%
Gastroenterostomy	Open ether	20 ozs.	60 mins.	38—38	No. est.	44
Gastroenterostomy	ditto	16 ,,	45 ,,	No est.—41	45	61
*Gastroenterostomy	Gas/oxy/ether endotracheal	3½ ,,	60 ,,	49—49	78	72
Appendicectomy and cæcopexy	Open ether	7 ,,	35 ,,	33—No est.	35	28
Goitre removal	Gas/oxy/ether endotracheal	2 ,,	55 ,,	20—20	No est.	23
Appendicectomy	Gas/oxy/ether face mask	1 ,,	20 ,,	No est.—26	33	30
Appendicectomy	ditto	4 ,,	45 ,,	18—20	21	24
Appendicectomy	Gas/oxy/ether face mask	2 ozs.	25 ,,	25—25	34	38
Appendicectomy	Gas and oxygen	No ether	25 ,,	33—35	35	35
Radium insertion.	ditto	No ether	20 ,,	No est.—20	35	30

*This case was found to have hyaline casts in the urine, and a blood urea of 62 mg.% ten days after operation. Probably a patient with some pre-operative kidney damage.

The blood-urea before operation was 38 mg. per 100 c.c., and it was estimated daily for four days, and then two days later. The following are the figures:—

41 mg.	per	100 c.c.	first	day	after	operation.
96 mg.	,,	,,	second	,,	,,	,,
138 mg.	,,	,,	third	,,	,,	,,
127 mg.	,,	,,	fourth	,,	,,	,,
96 mg.	,,	,,	sixth	,,	,,	,,

It is quite true that this patient did not suffer clinically from untoward symptoms. He was a strong, healthy man, aged 65 years, but it is manifest that the condition of the blood-urea following the operation was important, and under less favourable conditions might have been disastrous. It will be noticed, however, that the blood-urea before operation was within what are looked upon as normal limits.

The conclusions I draw from the foregoing biochemical investigation are as follows:—

(1) The higher limits of what is called normal blood-urea are abnormal. The higher the urea content of the patient before operation the greater the rise in the following few days.

(2) The cause of the increase is the effect of ether on the renal cells.

(3) In cases of enlarged prostate with a blood-urea approaching 40 mg. per cent, serious consideration should be given to the operation being performed in two stages.

(4) When there is an abnormal blood-urea and any renal damage, gas and oxygen anæsthesia only should be used.

References.

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2. Stephen, F. *Brit. Journ. Anæsth.*, 1929, vi, 109.

THE ASSOCIATION OF ANÆSTHETISTS.

WE are informed by the Secretary that the Council of the Association has met several times since our last report and has been busy with affairs concerning the welfare of anæsthetists. It has been consulted both by individual anæsthetists and by hospitals with regard to the arrangement of work and the position of anæsthetists at institutions. We are heartily glad to be able to record the recovery of the President, Dr. H. W. Featherstone, from the serious illness which laid him low at Christmas-time. Dr. Featherstone took the chair at the General Meeting, held on April 21st, at the room of the Medical Society of London in Chandos Street. Most of the Council and sixty members were present. Thirteen new members, whose names had passed the Council, were balloted for and elected; they came from Edinburgh, Manchester, Liverpool, Leicester, Oxford, and London. Dr. J. H. Chaldecott has resigned from the Council on retiring from the active staff of St. Mary's Hospital and Dr. C. W. Morris, of University College Hospital, has been elected in his place. The Secretary presented the report of consultations with the Specialists' Committee of the London County Council Consultants, with regard to the remuneration and status of anæsthetists in the L.C.C. general hospitals. A letter from Sir Frederick Menzies (Chief Medical Officer of the L.C.C.) was read, in which he stated that the fees of Anæsthetists were under review. The Secretary was instructed to write thanking Sir F. Menzies for bringing this matter to the notice of the Public Health Committee. Dr. Walter, of the Research Department of the Birmingham Corporation, gave to the meeting a very complete description of the manufacture and properties of the new light steel cylinders for carrying gas. His lecture was illustrated by lantern slides and was greatly appreciated.

Dr. Howard Jones (London) gave notice of a motion to be put to the Annual General Meeting in October: "That

this General Meeting of the Association of Anæsthetists of Great Britain and Ireland views with distrust any proposal to extend unqualified practice in anæsthetics as being in direct conflict with the aims of the Association. In view of the urgent demand for an efficient service of anæsthetics in obstetric work the meeting resolves to advocate the instruction examination and qualification of individuals in the best obstetric anæsthetic practice which the knowledge of the day provides."

BRONCHOSCOPY IN THE DIAGNOSIS AND TREATMENT OF POST-OPERATIVE LUNG COMPLICATIONS

By V. E. NEGUS, M.S., F.R.C.S.*

ALL surgeons and anæsthetists are interested in post-operative lung complications, and concerned as to their avoidance; particularly is this true for those who have to deal with operations in the upper air passages.

The nose and throat surgeon is called upon to diagnose and treat cases of lung suppuration with the bronchoscope; has opportunities of studying the normal mechanism of the tracheo-bronchial tree by direct observation, and of examining cases in which pathological changes have taken place.

I myself have had the opportunity of dealing with a certain number of cases, but many of the observations are gathered from the teaching of Chevalier Jackson. He has thrown light on the causation of suppurative diseases of the bronchi and lungs, and from his wide experience has drawn invaluable conclusions as to their treatment and prevention.

Scope of discussion. I presume that the conditions to be discussed are those directly concerning the anæsthetist and the bronchoscopist; they may be classified thus:

1. Inspiration of foreign body.
2. Diffuse suppurative bronchitis.
3. Lung abscess and bronchiectatic abscess.
4. Acute massive collapse or atelectasis.
5. Multiple bronchiectasis.

Natural defences of the lungs. It is expedient to review briefly the factors concerned in protecting the lungs against infection or the entrance of foreign bodies.

Every minute of our lives bacteria are entering the lungs without causing pathological changes;[15, 17] the numbers of bacteria are increased enormously in patients with dental,

*Abstract of a paper read before the Section of Anæsthetics, Royal Society of Medicine.

nasal or phryngeal sepsis. Very frequently foreign bodies are in situations without danger during normal conditions, although during anæsthesia similar circumstances might lead to an accident.

(*a*) *Protective closure of the larynx* does not offer any real safeguard against bacterial infection; its useful role is concerned with the exclusion of foreign bodies. Protective closure is carried out by the ary-epiglottic folds, which are drawn together and at the same time forwards against the cushion of the epiglottis. The larynx closes either voluntarily, if warning is given, or involuntarily if an intruding substance touches the mucous membrane of the lower pharynx or the margins of the laryngeal aperture.

Vountary closure cannot be maintained for long, because rising CO_2 concentration stimulates the respiratory centre and causes the larynx to open. In such an operation as dental extraction the patient may be forced to take an inspiration at the precise moment when a tooth drops down, with the unfortunate result that the foreign body is carried by the inspiratory air stream into the lungs.

During the induction of anæsthesia voluntary control is obliterated, and a deep inspiratory effect may suck in packing placed in the nose if it be not securely fastened.

The ease with which liquids will enter the larynx is seen in Singer's drop method of introducing lipiodol into the lungs. If the oil be poured drop by drop on to the base of the protuded tongue of a perfectly conscious individual it will enter the trachea.

Mullin[28] found that India ink placed in the nose of rabbits enters the lungs readily.

During anæsthesia liquids enter even more readily, as illustrated by the observations of Myerson[29] who passed a bronchoscope on patients after tonsillectomy. Blood was found in the trachea and bronchi in a large proportion of cases, namely in 76 out of 100 cases. Hara[11] examined 110 patients, after tonsillectomy and found blood in very many in greatest quantity after operation in the sitting position.

Similar conclusions were drawn by Benjamins[1] who

placed lipiodol on the tonsils before operation and saw it in the lungs by X-rays.

If the larynx be made insensitive with cocaine, blood-pus or foreign bodies will enter more readily still, either during local or general anæsthesia. It is not even necessary to spray, drop or paint cocaine solution on to the larynx. If any trickles down or is purposely applied to the hypopharynx it will cause block anæsthesia of the superior laryngeal nerves,[27] which supply the upper part of the pyriform fossa.

Anæsthetization of the larynx does not affect the cough reflex in the bronchi, however.[18]

The protection of the larynx is diminished or abolished by mechanical interference, noticeably by the use of the Davis gag, which lifts the tongue and epiglottis and may, in certain cases, prevent the ary-epiglittic folds from reaching the base of the epiglottis. Prevention of accumulation of blood by suction is the means of avoidance of danger under such circumstances.

(*b*) *Cough* is, of course, a most important method of defence if foreign bodies try to pass or have passed the larynx.

Sensory stimulation of the laryngeal, tracheal, or bronchial mucous membrane causes closure of the larynx accompanied by a forced expiratory effort. The ventricular bands, as well as the vocal cords, are closed, and a high pressure is reached. The larynx is then opened suddenly, and the escaping blast of air may remove the foreign body or infected secretions if they are not jammed in the bronchi. If a catheter be in position between the vocal cords the mechanism is inefficient, because the larynx cannot close completely.

In a few minutes, however, the sensitiveness of the mucous membrane disappears and no further effort is made to expel the intruding object or the exudate.[18] A solid body or a clot may lie in a bronchus for an indefinite period without inciting any cough after the first paroxysm has passed.

It is obviously fallacious, therefore, to rely on the cough reflex for protection of the lungs, because of its brief duration.

A second factor in cough must be considered. This is in connexion with the inspiratory opening and expiratory closure of the bronchi which occur during normal respiration;[4, 6, 15] both opening and closure are much more marked during cough, as may be seen clearly during bronchoscopy.[21]

Closure appears to be due to the active contraction of bronchial and bronchiolar muscles; inspiratory dilation is caused by elastic recoil of the bronchi, and the pull of the expanding lung which itself depends on the negative pressure in the pleural cavities.

Expiratory contraction squeezes secretions out of the bronchi, possibly in a peristaltic manner.

Any object entering the larynx during inspiration is sucked through the trachea into one or other widely open bronchus, and travels as far as the calibre allows. At expiration the bronchial walls contract on it and hold it firmly; the more violent the cough the more firmly is it held, particularly if it be of a shape to fit the bronchus, as for instance a tooth or a clot of blood or semi-solid secretion.

The distance to which an object travels down the tracheobronchial tree depends to a great extent on the depth of inspiration; the forced inspiration of the semi-conscious patient is liable to carry the intruding substance to a depth from which it cannot be removed by subsequent cough. In addition to changes in size during such respiratory cycle, the bronchi also show peristaltic waves travelling upwards, independent of cough, and of larger cycle than the respiratory movements. They have been described by Bullowa and Gottlieb.[5] These movements help to empty the lungs of secretion; they are much faster than ciliary action.

(*c*) *Secretion of mucus* is of great importance in preventing infection of the lungs. It is not necessarily bactericidal, but is strongly inhibitory to the growth of organisms.[9] One requisite for its efficient action would appear to be constant replacement; this necessitates satisfactory aeration of all parts of the lungs.

(*d*) *Ciliary action* is the last factor to be considered in studying the mechanism of protection of the lungs. It is specially valuable in removing bacteria before they obtain

a foothold; it is not sufficiently strong to remove a solid object, except one of minute size.

The cilia all wave in one direction, and a stream of mucus is carried along in the direction of their recoil.[34]

The cilia have no effect on secretion at a distance from them; if a bronchus becomes filled with secretion ciliary action will not remove it, because of back currents.[32, 33]

Cough can of course remove masses of secretion and small solid objects, but it is not the normal method of clearing away bacteria carried into the finer air passages by the inspiratory current.

It is on ciliary action that elimination of germs depends; it is most important in emptying the periphery of the lungs.[23] Efficiency of the action is influenced by several factors.

Cilia must be bathed in mucus, and this mucus must be of a faintly alkaline reaction; many observers have noted that weak acids paralyse cilia.[2] Leonard Hill[14] has measured the rapidity of action accurately. I have seen immediate paralysis with a solution of acetic acid 1 in 20.000. CO_2 is strongly inhibitory because it makes the secretions acid; the action is, however, no more than temporary.

Alterations in the *p*H of the tissue fluids appear to affect the action and, therefore, acidosis is to be avoided. Prolonged administration of both ether and chloroform reduce the alkali reserve of the blood.[13]

The end products of some pathogenic organisms have an acid reaction, as noted in suppuration of nasal sinuses by Mittamaier,[26] and therefore a vicious circle is brought about by bacterial activity in the bronchi, as for instance after breaking down of an inspired and infected clot. The products of growth paralyse the chief means of removal of the cause of disease. Ether vapour is temporarily inhibitory and chloroform much more powerfully so. Coal-gas stops cilia.[14]

Proetz has, however, seen active ciliary movement on pieces of mucous membrane removed from the nose of patients suffering from suppurative rhinitis.[33]

Interruption of ciliary action is caused if liquid chloroform

or ether lie on the mucous membrane, as observed by Professor Hill.[14]

If ether be warmed so much that it boils it will give off vapour which may re-condense in the delivery tube and even reach the trachea or bronchi in liquid form.

In the bronchiactatic abscess—a condition particularly liable, according to Lilienthal,[25] to follow the operation of tonsillectomy under general anæsthesia—the lining membrane is covered with stratified squamous epithelium and not by columnar ciliated type. Ciliary action is not available, therefore, to empty the cavity.

THE RESULTS OF INEFFICIENT DEFENCE OF THE LUNGS.

1. *Inspiration of a foreign body* may occur during general or local anæsthesia. The objects likely to enter the lungs are teeth, pieces of swab or sponge,[38] a part of a tonsil, adenoids, or a nasal plug.

During dental extraction of teeth with local anæsthesia it has happened many times that a tooth has slipped from the grasp of the forceps, or that the crown has broken off and fallen down the pharynx, while the patient gasped—not unnaturally—when taken unawares.

With general anæsthesia the danger of entrance of a foreign body is greater if the operation is performed in the sitting position.

The administration of gas eliminates the voluntary effort of the patient to close the larynx, while still allowing deep inspirations to be taken. It is not surprising, therefore, that teeth are inspired. Safety is attained by protecting the pharynx with a sponge, or by performing the operation in the lying position with the use of a head-light.

During tonsillectomy the greatest danger of inspiration of foreign bodies occurs when the patient is sitting up, for a dislodged tooth or a piece of sponge easily follows the air stream down the trachea.

Bronchoscopy is, of course, the only method of removal of foreign bodies. Every object which passes down through the glottis can be removed by the same route, as taught by Jackson.

Diagnosis is made on the history of the accident on symptoms of cough followed by expectoration, and on the physical and X-ray signs.

If suspicion of a foreign body is aroused, it is best to disprove or confirm the diagnosis by the use of the bronchoscope with local or general anæsthesia.

I have at 6 o'clock removed a tooth which was lost in the patient's mouth at 4.15; it showed up in a radiograph unlike some others which are not easily seen.

It is well to realize that the entrance of a foreign body is an easy occurrence; much valuable time would be saved if bronchoscopy were resorted to early.

2. *Diffuse suppurative bronchitis* may arise after general anæsthesia; it is characterized by the expectoration of copious purulent sputum.

The accidental entrance of liquid ether or chloroform will cause it: if such an accident occurs it is quite possible that bronchoscopic lavage, performed early, would save the patient from death. A suction pump may be connected to an endotracheal catheter in such cases, if a bronchoscope is not available.

Complete paralysis of ciliary action is an important factor in this condition, the anæsthetic remaining in the tracheobronchial tree, and there causing acute inflammation. A vegetable foreign body inhaled into the trachea or brochi produces similar symptoms; the entrance of such an object is not, however, likely to be associated with anæsthesia. Many common causes are the entrance of infected blood, of pus, or of vomit. Infection is very liable to occur if dental sepsis is present.

Bronchoscopy, in my opinion, is of great value in cases of severe post-operative suppuration. Removal of the pus by aspiration may prevent the patient from drowning in his own secretions, as described by Chevalier Jackson. If a tracheal cannula is in position, a rubber catheter may be passed through it and secretions aspirated by means of an electric suction pump kept at the patient's bed side. Ciliary action is insufficient to remove copious secretions; it may even be paralysed by the acid reaction of the sputum. Cough

does no more, in many instances, than propel the sputum half-way up the trachea, whence it gravitates once more into the bronchi. This is well seen with a bronchoscope, for even though a patient has coughed violently before the introduction of the tube, yet his bronchi may be found almost full of pus.

The bronchoscope also allows one to see that when the bronchial walls are inflamed they come together completely during cough, instead of merely causing diminution of the bronchial lumen.

The result is that any pus in the alveoli or finer air tubes beyond the inflamed bronchi cannot escape. It is necessary to insert an aspirating bronchoscope to hold open the inflamed walls, when cough will be able to expel secretions from the distal area, or to remove the pus directly by means of a fine flexible suction tube.

3. *Lung abscess and bronchiectatic abscess.* The most frequent cause of these conditions is the inspiration of infected blood-clot or an infected foreign body such as a tooth or a swab, or vomited material, during operations on the mouth or pharynx, and in particular during dental extraction and tonsillectomy. Pulmonary abscess is said to occur once in every 2,500 to 3,000 tonsillectomies.[27] Entrance of blood or pus alone may cause bronchitis, but a lung abscess is not likely to appear unless a bronchus becomes partially or completely blocked.

A clot from the mouth or pharynx passes the larynx either because the latter is held open by a Davis gag, or because deep inspirations are taken during the operation, or because the latter is performed in the sitting position, and particularly if the hypo-pharynx has been anæsthetized, as may occur during tonsillectomy with local anæsthesia. The clot descends with bacteria caught up in it; it is sucked down as far as its size permits, and cannot be expelled by cough because of expiratory contraction of the bronchi. No inhibitory mucus can reach the entangled bacteria, and ciliary action is not sufficiently powerful to dislodge the mass.

The bacteria grow and multiply and cause inflammatory swelling in the bronchial walls, often with the shedding of

epithelium, and later with formation of granulations. Because of the obstruction, mucus cannot escape from distal bronchi, and it is, therefore, not replenished; bacteria grow and cause infection of a lobe. Air cannot enter, and that which is present is absorbed, with collapse of the alveoli.

When the clot disintegrates there may be so much swelling of the bronchial walls that entrance of air and escape of secretions are still prevented. Ciliary action is ineffective because of mechanical obstruction in the passage of secretions, or because of the formation of granulations, or the paralysing action of pus. Cough may raise the pressure of retained air or of secretions beyond the obstruction, and may thus blow out the weakened bronchial walls just distal to the point of blockage, with formation of bronchiectasis. Cavities in the lungs due to local suppuration may be an abscess in the parenchyma of the lungs, draining into a bronchus, or a localized dilatation of a bronchus, namely a bronchiectatic abscess.

The patient develops symptoms suggestive of lung abscess on about the seventh or tenth day; the diagnosis is confirmed by the physical and X-ray examination.

Bronchoscopy should not be held in reserve; its early use may be the means of avoiding a long illness or of saving the patient's life. If a patient coughs up large quantities of pus during a short period of time, that is sufficient evidence that he has a reservoir of infected secretions; as in every other abscess in the body, the most important point is to establish drainage. This is done by bronchoscope with local anæsthesia. An aspirating bronchoscope is passed and the diagnosis is confirmed, if necessary, by locating the escape of pus from one lobe. Swelling of bronchial walls and the formation of granulations are visible. If no pus escapes a flexible suction tube is passed into the bronchus leading to the suspected lobe, and immediately, or subsequently, pus may be expelled from an unplugged tube.

When the site of the abscess is located aspiration is used to remove infected clot and a flexible tube is passed to reopen stenosed bronchi; granulations are painted with 10 per cent solution of silver nitrate.

The procedure may need repetition on one or more subsequent occasions. Chevalier Jackson[19] reported cure in 61 per cent in a series of 224 cases following tonsillectomy. If a foreign body is discovered and removed, rapid cure is certain to follow. It has been thought that many lung abscesses are embolic in origin because they occur in the upper lobe of the right or left lung.[30] I do not think this to be a valid argument.

If a patient is lying flat or is sitting up, a clot, like any other foreign body, may travel straight down into the lower lobe, more often on the right side, because that bronchus is more in line with the trachea than is the left branch. If, however, the patient has returned to bed and is lying on the side when a clot is carried in by a powerful inspiratory effort, it is probable that the object will fall into the open mouth of the upper lobe bronchus over which it must pass; the force of gravity determines its direction to a certain extent. The inspiratory current of air may, of course, carry a light object, such as a clot, into an upper lobe bronchus with great ease, even if the patient is lying flat. Some observers report that post-operative lung abscesses occur more often in the upper than in the lower lobes.[10]

There is no doubt that some abscesses form in the lung parenchyma and later burst into a bronchus, but it is probable that the number is few; of those thought to arise in this way, because of no obvious communication with the bronchus when they are first diagnosed, a certain number no doubt commence in a small bronchus which becomes blocked by the infected clot which causes them.

4. *Acute massive collapse of a lobe* sometimes calls for bronchoscopic diagnosis and treatment. The observations of bronchoscopists and particularly those of Chevalier Jackson and Gabriel Tucker[39] show that in many cases the condition is caused by plugging of a bronchus with viscid mucus.[12] Aeration of the lung beyond the obstruction is impossible, and the air in the shut off lobe is absorbed, causing atalectasis.[8] For the reasons already given lack of fresh supplies of mucus and absence of ciliary action allow bacteria to grow and multiply. Inflammatory reaction causes still greater

obstruction and catarrhal or purulent exudation. The affected lung area is thus drowned in secretions.

Bristoe has shown that deflation is a common post-operative occurrence[4]. Absence of air and absence of respiratory movements in a lower lobe favour stagnation of mucus. The patient suffers from fever and coughs, and appears to be very ill. There is deficient or absent entry in the shut off area and upward displacement of that side of the diaphragm.[7, 3] The heart is displaced towards the affected side.[22]

Massive collapse may occur after any operation, but particularly after those on the upper abdomen; deficient movement of the diaphragm as originally described by Pasteur[31] is no doubt a very powerful factor associated with deflation.[4] Inspiratory dilation of bronchi and bronchioles is diminished and secretions are not removed.

If suspicion arises that a bronchus is blocked, bronchoscopic examination is called for, if medical treatment, with correction of inefficient breathing, does not cure the condition rapidly by producing expansion of the lung.

The instrument is passed, preferably with local anæsthesia, and may reveal redness and swelling of a lower lobe branch, possibly with complete occlusion; probably little pus may be seen, because of bronchial obstruction. Passage of a fine flexible-ended suction tube down the suspected bronchus may release purulent secretion, either immediately or after aspiration.

The patient will cough up much purulent sputum, and his symptoms may improve rapidly even after one bronchoscopic treatment.

5. *Multiple bronchiectasis* sometimes dates from the time of an operation. The history, however, is often so long that it is difficult to say whether the cause was in the operation itself or in the disease it was intended to relieve. For instance many cases follow suppurative sinusitis, the lung symptoms and signs commencing after the operation. It is probable, however, that infection from the patient's own pus is the cause. This supposition is supported by the fact that

many cases commence in childhood before any operation has been performed.

Diagnostic bronchoscopy reveals escape of pus from one or more branch bronchi, often with redness of the walls and the formation of granulation.

Aspiration of pus, destruction of granulations by swabbing with 10 per cent solution of silver nitrate and dilatation of stenosed bronchi may give considerable relief. The condition is, however, difficult to treat, and in most cases nothing more than alleviation of symptoms can be obtained.

Avoidance of Lung Complications.

1. *Prevention of inhalation of a foreign body* is difficult if operations are performed in the sitting position. Protecting the larynx by means of a sponge in the pharynx during tooth extraction is a necessary precaution. In tonsillectomy cocainization of the larynx should be avoided by seeing that no cocaine trickles down to the hypopharynx. With general anæsthesia for operations in the mouth or throat safety is assured by the use of the Rose or Trendelenburg position and by the prevention of deep inspirations. Loose objects will tend to drop into the pharynx and not into the larynx, if the head be lower than the body.

Nasal packing must be anchored outside the nose, the gauze in the nasal fossæ being tied together: there should be a single piece only of plugging in each side of the nose. Plugs—particularly those made from glove fingers—put in after such operations as submucous resection must be tethered with threads and strapping.

2. *Diffuse suppurative bronchitis* caused by entrance of blood or pus is not likely to happen in nasal operations with local anæsthesia, because secretions are swallowed and not inhaled. In tonsillectomy in the sitting position care must be taken to avoid anæsthetization of the larynx.

Dental sepsis must be attended to before operation in all cases.

When general anæsthesia is employed the dangers are considerable.

During dental extractions the pharynx must be protected by gauze or sponge.

In operating on the nose and sinuses safety is attained by the use of endotracheal anæsthesia with return airway, the pharynx being packed with gauze, as advocated by Magill. It would appear unwise to introduce a catheter through the nose into the larynx, if nasal sepsis is present.

In operating on the mouth or pharynx in adults, endotracheal anæsthesia is a safeguard. The respirations should be kept so shallow that no secretion is sucked in through the larynx. The patient should be in the Rose or Trendelenburg position. Suction is essential, preferably by means of a gum elastic catheter passed through the nostril into the nasopharynx; the latter acts as a sump. It is, in my opinion, inadvisable to anæsthetize the laryngeal aperture, because of the danger of inefficient closure when the patient returns to bed. He should have the foot of the bed raised and should lie on his side, particularly if peraldehyde or avertin have been given beforehand; little danger should arise if these precautions are taken.

In paryngeal operations tracheotomy is of great use, a window being made and a pack of gauze being inserted through it from below upwards towards the larynx. An excellent form of anæsthesia in my experience is rectal oil-ether, followed by a little chloroform.

After operation rapid recovery of the patient from the anæsthetic is desirable and the sitting position is used, so that free breathing and effective cough may expel secretions.

Over-use of drugs likely to diminish cough, such as morphia, is avoided. Morphia is said also to slow ciliary action.[14]

Condensation of ether in the bronchi is naturally to be avoided. Such an accident appears to be a real possibility; it will not occur unless the ether bottle is overheated. CO_2 given at the end of the anæsthetic readjusts the composition of the alveolar air and of the blood and tissue fluids.[41] Any inhibitory effect of this gas on ciliary action passes off quickly.[14]

As regards the use of atropine, it would appear that if so much were given that the bronchial mucuous membrane became quite dry, there would be paralysis of ciliary action and loss of the inhibitory power of mucus, with the probability of growth of bacteria. Such an occurrence is not likely because of the irritative effect of anæsthetic vapours. Even with the biggest doses of atropine in use there is an excess of secretion.

3. *Prevention of lung abscess and bronchiectatic abscess* follows the same lines as those just enumerated. In addition it is important to prevent the formation of clots; this is effected by seeing that blood does not accumulate in the nose, nasopharynx and pharynx during operation, and that it is all removed before the return of the patient to bed.

It is quite possible, if precaution is not taken, to find inspiration of clots. The field of operation may have appeared quite dry, but blood may have been left behind, particularly in the nasopharynx, in which it is hidden, to descend when the patient changes position on return to bed.

If clots are present in the mouth, nasopharynx, or pharynx at the end of operation, the use of CO_2 may be dangerous because the forced inspirations may suck them into the trachea or bronchi.

4. Free aeration of the lungs is necessary to prevent solidification of mucus in a bronchus causing *massive collapse*. The use of CO_2 at the end of operation appears to meet this end with the precaution mentioned above; temporary paralysis of cilia may be produced, but this soon passes off when oxygen or fresh air reaches the bronchi.

Paralysis of cilia after descent of liquid chloroform or ether is not likely to occur, except under most unusual circumstances.

After operation, if possible the patient should be placed in such a position that the bases of both lungs can expand; a permanent position on one side may lead to trouble. After laryngeal operations blockage of a bronchus is prevented by the use of suction through the tracheotomy cannula.

5. The avoidance of *bronchiectasis* is attained by taking the precautions suggested under the headings of suppurative bronchitis. Multiple dilation of bronchi may follow long standing inflammation of the bronchi, with blowing out of the weakened walls during cough.

If measures are taken to prevent infection of the lungs at the time of operation, bronchiectasis will not be a complication of anæsthesia.

Summary.

Lung complications calling for bronchoscopy are not frequent because suitable precautions are taken by most anæsthetists and surgeons.

A certain number of cases occur, however, sometimes diagnosed as pneumonia, but actually due to mechanical obstruction of a bronchus.

Suspected cases require bronchoscopy for diagnosis, and respond well to treatment by aspiration and direct medication.

All cases due to foreign body can be cured, and most operative lung abscesses clear up quickly.

Diffuse bronchitis is improved and its cessation prevents the subsequent development of multiple bronchiectasis.

Acute massive collapse is in very many instances the result of occlusion of a bronchus by a plug of thick mucus, removal of which effects a cure.

References.

(This list is not comprehensive, but includes only papers referred to above.)

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ABSTRACTS.

Sodium soneryl as basal hypnotic. E. BIRDSALL. *British Medical Journal,* Aug. 20, p. 871.

The author writes from an experience of 150 cases at St. Bartholomew's Hospital. Sodium soneryl is the sodium salt of butyl-ethyl barbituric acid and is therefore closely allied to nembutal, but it is less toxic and is given in greater doses. The operations for which it was used were of various kinds, throat, abdominal, bone, etc., and the ages of the patients ranged from six to 70 years. The drug was given by the mouth in capsules containing 0.15 gr. Two capsules at 8 p.m. ensured a good night before operation. On the day of operation a dose was given one hour before operation, varying with the body weight of the patient, starting with three capsules for those who weighed from 6 to 8½ stone and reaching six capsules for those above 12½ stone. Later the dosage was simplified and the evening dose omitted. Patients under 10 stone got 0.6 gr., between 10 and 12 stone 0.75 gr., and above 12 stone 0.9 gr.

In 97 cases the patient was asleep on entering the operating theatre, in 45 sleepy, in five unaffected, and in two excited. No stertor or cyanosis in any instance. Sleep was generally produced in about half an hour. In many cases gas oxygen was used in addition. When ether was needed the amounts required were small. Recovery occurred in about three hours. Restlessness was observed in several instances (about 10 per cent of all). Amnesia was noticeable. Sodium soneryl is rgarded as a reliable basal narcotic when given by the mouth an hour before operation in doses of from 0.6 to 0.9 gr., according to the patient's weight.

Spinal peridural anæsthesia. PROF. A. M. DOGLIOTTI. *Anæsth. and Analg.,* March–April, 1933, p. 59.

A new method is described which is, in the author's opinion, able to give for the upper and lower abdomen, the extremities, and the thorax an anæsthesia comparable with that which epidural general anæsthesia provides for the perineum and the pelvis. Injection is made into the peridural space at any level, lumbar or dorsal, according to the region of the body which it is desired to render insensitive. The peridural space is found in the vertebral canal, between the bony structure and the dura mater, and continues upwards as far as the occipital foramen, where it ends because the dura adheres there to the periosteum. The space continues downwards with the sacral canal, laterally with the inter-vertebral foramina and it communicates with the para-vertebral tissue. It is occupied by loose connective

tissue, rich in blood-vessels, which are easily displaced. It is difficult, the author says, even with increased doses of solution, to make this reach the upper cervical or lower sacral regions. On one occasion after excessive injection he was obliged to use artificial respiration, but states that even in this instance the blood pressure did not fall notably. The anæsthetic solution acts on the spinal nerves extradurally and in the inter-vertebral foramina. It acts also on the spinal ganglia, rami communicantes, and the paravertebral sympathetics; only a very small portion passes through the dura mater into the cerebrospinal fluid. Consequently one can anæsthetize a series of spinal nerves without danger of diffusion towards the bulb, of serious fall of blood pressure, or of meningeal irritation. A deep and complete anæthesia of one segment of the body can be obtained without involving the parts below and above it. The difficulty of technique is overcome by introducing the needle, connected with a syringe filled with physiological solution, slowly and exercising a constant pressure on the piston while the needle penetrates the yellow ligament. During this time a strong resistance is felt to the injection of the liquid. As soon as the needle has pierced the ligament and arrived at the peridural space resistance disappears and the liquid enters with facility, separating the dura mater from the peridural adipose tissue. The needle is now in the desired space, and having ascertained that there is no flow either of blood or of cerebrospinal fluid the next procedure is the injection of the anæsthetic solution. This is a 1.5 per cent. solution of novocain in simple salt solution with drops of adrenalin. The injection is made slowly, five to ten minutes. From 30 to 50 c.c. are an average amount needed for an abdominal operation. The needle is introduced at the side of the spinous process, avoiding the interspinous ligament, on the side on which the wider anæsthesia is desired.

Pre-operative procedure. E. E. Flint. *Lancet,* June 23, 1933, p. 1165.

The author is " convinced that the safety of our patients lies more with improvements in pre-operative measures framed to overcome or prevent physico-chemical disturbances than in actual operative technique. If we operate without investigating and correcting these factors as far as possible there is the gravest danger of precipitating a series of phenomena which can be included under the title of secondary shock, phenomena much more easily prevented than cured. . . ." An effect of ether is to produce hyperglycæmia; in a liver somewhat depleted of its glycogen this additional attack may be serious. Another disturbing element is its solvent action on fats. Prolonged inhalation of ether has been shown to cause definite increase in fat content of the blood. This fat undergoes rapid metabolism in the presence of sugar, but deficiency in this substance results in more or less acidosis.

With reference to the above the reader may be recommended to see also *Archives of Surgery*, May 1933, vol. 26, No. 5, p. 909., where Hosper writes on blood cholesterol in anæsthesia. After discussing literature on the subject and describing experimental observations, the author's conclusions are:

(1) "Continuous ether anæsthesia in dogs and rabbits is accompanied by hypercholesteræmia that in ordinary surgical anæsthesia reaches a peak in from sixty to ninety minutes and thereafter greatly diminishes in amount, returning to approximately normal limits in from four to five hours.

(2) Continuous chloroform anæsthesia is accompanied by slight immediate change but by a gradual and continuous rise in blood cholesterol proportional to the duration of anæsthesia (up to 10 hours).

(3) Ethylene and nitrous oxide are accompanied by no change in blood cholesterol other than those explainable by excitement and imperfect anæsthesia.

(4) Excitement is accompanied by hypercholesteræmia. This factor increases, but is not entirely responsible for, the change found under ether anæsthesia. It seems to be responsible for initial changes under chloroform and for the irregularity in the blood cholesterol values under ethylene and nitrous oxide.

(5) Ether anæsthesia is also accompanied by a hyperglycæmia that frequently tends to be proportional to the hypercholesteræmia. Chloroform ethylene and nitrous oxide anæsthesia are accompanied by a hyperglycæmia as is excitement without anæsthesia.

(6) Ethylene and nitrous oxide, as anæsthetics, seem to be more physiologic in their action than ether and chloroform." [Exact meaning of this?—Ed.]

Explosibility of nitrous oxide-ether mixtures. L. F. Sise. *New England Journ. Med.*, May 4, 1933.

Attention is drawn once more to the explosibility, often insufficiently realized, of mixtures of ether with nitrous oxide and oxygen. Several instances are recorded, in one of which the anæsthetist was so severely burnt that he lost his life. The late Professor Dixon in this country, who did so much valuable work on explosives, drew especial attention to the possible dangers of this particular mixture of gases, but we are less exposed to the risk of static sparks to start the explosion than they are in America, owing to the greater general humidity of our atmosphere. Even the oxygen of the mixture is not necessary for explosion, as oxygen for the combustion of ether is readily derived from the breaking down of nitrous oxide into nitrogen and oxygen. With ordinary methods of administration explosive mixtures cannot be demonstrated more than a few feet from the mask. With CO_2 absorption the distance is reduced and with, in addition, a perfect fit of mask, ignition can take place only from within. The

greatest danger is from static spark and the best protection against this is humidity of the operating room.

Spinal anæsthesia. R. M. TOVELL, *Canadian Med. Journ.*, No. 4, April, 1933.

Patients whose blood pressure is extremely high generally experience marked reactions following spinal anæsthesia, and for them the dose must be conservative. Patients with blood pressure of 100 mm. of mercury or less should not be subjected to the method for operations above the lower part of the abdomen, unless a general anæsthetic is contra-indicated. Hæmorrhage in association with spinal anæsthesia is much more likely to cause shock than it is with ether. The method is therefore inadvisable when the patient has lost much blood or such loss is likely at operation. Ephedrine in a dose not exceeding 75 mg. is given before the injection or immediately after this. The dose of novocain to be injected is gauged roughly at 1 mg. to 1.5 mg. per pound of body weight. Control of duration and of upward extension of anæsthesia is obtained by varying the dose of novocain and the amount of spinal fluid with which it is diluted. Two hundred mg. are generally enough for operations on gall-bladder or stomach. If one is limited to 200 mg. of novocain, which should rarely be exceeded, the duration of the anæsthesia is often not enough. Various drugs have been tried to get over this difficulty, including percaine, the action of which, however, was considered to be dangerously prolonged. A combination of 5 mg. or 10 mg. pantocain with 50 mg. to 100 mg. novocain is found to give the desired result. The duration of anæsthesia is from one and a half to twice as long as that obtained from a safe dose of novocain alone for the same case. Pantocain is a derivative of novocain, and is about nine times as potent. For this reason, and because it fails to arrive at a fixed position in relation to the nerve roots to be attacked as quickly as does novocain, it is reserved for operations below the umbilicus. Patients under spinal anæsthesia have occasionally been found to complain of pain in the legs, although at the time anæsthetized and relaxed to the costal margin. Supplementary anæsthesia with nitrous oxide or ethylene should then be employed.

Synergism between magnesium salts and ether. L. F. SHACKELLAND and R. R. BLUMENTHAL. *Anæsth. and Analg.*, May–June, 1933.

The Gwathmey technique of obstetric anæsthesia, as is well known, calls for intramuscular injections of magnesium sulphate. The total quantity administered is about 0.06 grm. per kilo of body weight, and the minimal effective depressant dose for human beings is about four times that amount. The theory, however, on which Gwathmey bases his use of magnesium sulphate is that it synergizes the anæsthetic action of the ether. Numerous experiments carried out by the writer

fail to show any action by magnesium sulphate of the kind claimed by Gwathmey. Some of his conclusions are: (1) A simple summation of anæsthetic actions as well as of toxicities were obtained with regularity when various mixtures of magnesium sulphate and ether were administered to rats by the vein. (2) The narrow margin of safety seen with magnesium sulphate alone was preserved in its combination with ether; in other words, the margin of safety for ether was narrowed by combining it with magnesium sulphate. (3) There is no evidence in support of the use of magnesium sulphate or other salt of magnesium as an adjunct to ether oil anæsthesia.

In the same journal (page 113) L. G. Reynolds discusses the choice of anæsthetics for prostastectomy. His favourite procedure is spinal anæsthesia obtained by 100 mg. novocain crystals in 2 c.c. of spinal fluid. He uses the lowest interspace that can be easily punctured. At 9 p.m. on the day before operation 3 gr. sodium amytal are given by the mouth. This is repeated at 7 a.m. on the morning of operation, and half an hour before going to operation omnipon gr. ⅓ is given hypodermically. Just before the spinal puncture ¼ gr. of ephedrine are given subcutaneously.